AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 110:351–363 (1999)

Enamel Hypoplasia in Deciduous Teeth of Great Apes: Do Differences in Defect Prevalence Imply Differential Levels of Physiological Stress?

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KEY WORDS enamel hypoplasia; enamel defect; localized hypoplasia; deciduous teeth; hominoids; great apes

ABSTRACT This paper presents new data on enamel hypoplasia in the deciduous canine teeth of great apes. The enamel defect under consideration is known as localized hypoplasia of primary canines (LHPC), and is characterized by an area of thin or missing enamel on the labial surface of deciduous canine teeth (Skinner [1986a] Am. J. Phys. Anthropol. *69:*59–69). Goals of this study are: 1) to determine if significant differences in the frequency of LHPC occur among three genera of great apes, and 2) to evaluate variation in LHPC prevalence among great apes as evidence of differential physiological stress. Infant and juvenile apes with deciduous teeth were examined at the Cleveland Museum of Natural History (n = 100) and at the Smithsonian Institution, National Museum of Natural History (n = 36). Deciduous teeth were observed under oblique incandescent light, with the naked eye and with a 10× hand lens. Enamel hypoplasia was scored using Federation Dentaire International (FDI)–Defects of Dental Enamel (DDE) standards. Hypoplasias were recorded by drawing defect location and size on a dental chart, and by measuring defect size and location with Helios needlepoint dial calipers.

The prevalence of LHPC is reported by genus and sex, using two approaches: 1) the frequency of affected individuals—those having one or more deciduous canine teeth scored positive for LHPC; and 2) the number of canine teeth scored positive for LHPC as a percentage of all canine teeth observed. Variation in defect size and location will be described elsewhere.

Localized hypoplasia of primary canine teeth was found in 62.5% of 128 individual apes, and in 45.5% of 398 great ape deciduous canines. As in humans, LHPC is the most common form of enamel hypoplasia in deciduous teeth of great apes, while LEH is rare or absent. The distribution and pattern of expression of LHPC in great apes is similar to that described in humans: side differences are not significant, but mandibular canines exhibit the defect two to five times more often than maxillary canine teeth. Differences in LHPC prevalence by sex are small and not significant. Intergeneric differences are large and non-random: chimpanzees (*Pan*) exhibit a significantly lower frequency of LHPC (22%, n = 50) by individual count, than either the orangutan (*Pongo,* 88.0%, n = 25) or the gorilla (*Gorilla,* 88.7%, n = 53). Tooth count prevalences exhibit a similar pattern of variation and are also statistically significant. These findings suggest that large bodied great apes (gorilla

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Received 3 December 1998; accepted 20 July 1999.

and orangutan) may be under greater physiological stress during perinatal and early postnatal development than the chimpanzee. The size, position, and timing of LHPC lesions are currently under analysis and may yield more insight into the etiological origin of this enamel defect. Am J Phys Anthropol 110:351–363, 1999.

Studies of enamel hypoplasia in the deciduous teeth of great apes are rare, of a preliminary nature, and based on small samples. The purpose of this paper is to present new data on the prevalence and distribution of enamel hypoplasia in the deciduous teeth of great apes and to interpret the results within the context of a generalized physiological stress model. A particular form of enamel hypoplasia, often referred to as localized hypoplasia of primary canines (LHPC; Skinner 1986a), is the subject of this report (Fig. 1). LHPC is easily differentiated from the more commonly discussed enamel defect known as linear enamel hypoplasia (LEH) by several criteria: 1) form of expression (non-linear), 2) distribution in the dental arcade (restricted to deciduous canine teeth), and 3) location on the dental crown (labial surface) (Lukacs and Walimbe, 1998, 1999). In LEH one or more horizontal grooves or a linear array of pits representing a deficiency of enamel formation are present on the outer enamel surface (Goodman and Rose, 1990, 1991; Hillson and Bond, 1997). LEH is more frequently observed in permanent than in deciduous teeth, and adjacent teeth are often affected. Matching LEH defects are often discernable on antimeric pairs and on dental crowns segments developing simultaneously, thus constituting a nonspecific indicator of systemic growth disruption (Goodman and Rose, 1990; Skinner and Goodman, 1992). The clear distinction in the appearance of these two types of enamel defects, and their dissimilar distribution in the dental arcade suggest that significantly different etiological pathways are involved.

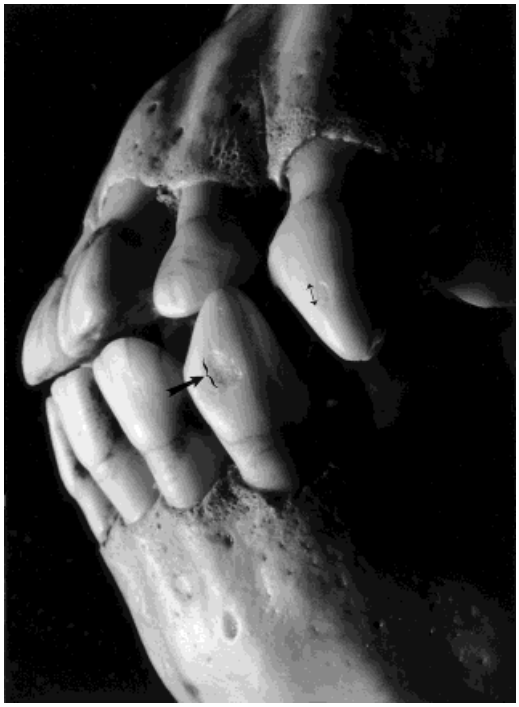

Fig. 1. Specimen of a juvenile orangutan showing characteristic appearance of LHPC (arrows).

Several recent studies of great ape dental pathology provide valuable new data on pathological lesions affecting permanent teeth, but developmental enamel defects and observations on deciduous teeth were not recorded. The focus of these studies was to document degenerative dental afflictions (such as dental caries, periodontal disease, abscesses and antemortem tooth loss, for example) of permanent teeth (Lovell, 1990a, 1991). Skeletal and dental pathology of adult free-ranging mountain gorillas (*Gorilla gorilla beringei*) housed in the National Museum of Natural History were documented by Lovell (1990b). Reports on the dental pathology of orangutans (*Pongo satyrus borneensis*; Stoner, 1995) and of free-ranging chimpanzees (*Pan troglodytes schweinfurthii*) from Gombe National Park (Kilgore, 1989) do not contain data on enamel hypoplasia prevalence or observations of deciduous dental pathology.

Prevalence and severity of physiological stress during permanent dental development in non-human primates has been inferred by several investigators using the

incidence and prominence of linear enamel hypoplasia (LEH) (Jones and Cave, 1960; Miles and Grigson, 1990; Moggi-Cecchi and Crovella, 1991; Skinner, 1986b; Skinner and Guatelli-Steinberg, 1997; Vitzthum and Wikander, 1988). Problems common to many of these studies are small sample sizes and the practice of combining observations from several species, thus obscuring species-specific patterns of prevalence and distribution. By contrast, a recent study of LEH in 360 rhesus macaques from Cayo Santiago revealed a previously undescribed pattern of expression in which linear defects occurred most often in the sectorial premolar (Guatelli-Steinberg and Lukacs, 1998). A further limitation of the analyses of LEH in non-human primates is that few document prevalence of enamel defects in the deciduous teeth, despite the unique insights to be gained regarding physiological stress during prenatal and perinatal development.

Since some deciduous dental enamel forms prenatally, prevalence of defective or hypoplastic enamel may serve as a retrospective measure of non-specific physiological stress during the earliest stages of development. Do the three genera of great apes in this study (*Gorilla, Pan,* and *Pongo*) exhibit similar frequencies of defective enamel in their deciduous canine teeth? If not, can differences in prevalence of enamel hypoplasia be interpreted to indicate differential levels of developmental stress among the great apes? Being the first comprehensive analysis of enamel hypoplasia in great ape deciduous teeth, the research findings reported here have significant implications for understanding variation in perinatal and infant physiological stress among modern great apes and for enhancing knowledge of the defect's range of expression in comparison with human populations.

While copious literature is available documenting prevalence and etiology of enamel hypoplasia in human populations in clinical, epidemiological, historic, and prehistoric samples (Goodman and Capasso, 1992; Goodman and Rose, 1990, 1991; Skinner and Goodman, 1992), far less is known about the prevalence and distribution of enamel defects in the permanent teeth of non-human primates, living or fossil. A synoptic historical review of the analysis of enamel hypoplasias in non-human primates is provided by Eckhardt (1992).

Studies of enamel hypoplasia in the *deciduous* teeth of great apes are rare, of a preliminary nature, and based on small samples. Deciduous teeth were excluded from Skinner's investigation of enamel hypoplasia among 229 great ape specimens from sympatric populations of *Pan* and *Gorilla,* because, "Deciduous teeth . . . are relatively free of defects"(Skinner, 1986b:294). In an analysis of linear enamel hypoplasia in *Pan troglodytes* from Liberia, Eckhardt (1992) reports observations for deciduous maxillary incisors and for deciduous mandibular canine teeth: "In the case of the maxillary central incisor, 12 of 70 specimens had deciduous teeth present, *none of which exhibited transverse enamel hypoplasia*" and "For the mandibular canine, 20 of the 59 specimens had deciduous teeth present, *none of which exhibited enamel hypoplasia* (emphasis mine)."

Since the focus of both Eckhardt's and Skinner's research was linear enamel hypoplasia (LEH; also known as transverse enamel hypoplasia), their observations can be regarded as consistent with regard to this type of enamel hypoplasia. Less clear, and somewhat perplexing, is the lack of comment on the presence or absence of localized enamel hypoplasia of primary canine (LHPC) teeth in these study samples because in the same year Skinner (1986a) published the first extensive analysis of localized enamel hypoplasia in human deciduous teeth.

The only study of enamel hypoplasia exclusively devoted to deciduous teeth of great apes is by Eckhardt and Protsch von Zieten (1993). They report individual count prevalence of enamel hypoplasia in a sample of 70 specimens housed at the Institute of Anthropology and Human Genetics, Goethe University, Frankfurt am Main, Germany. Positive aspects of this study sample are that it derives from a free-ranging, natural population, and is of respectable size. Limitations include the high degree of postmortem damage to jaws and teeth, the extensive postmortem loss of teeth, and the fact that crania often lack mandibles. The conclusions of this investigation were: 1) that the deciduous type of hypoplastic defect observed was "pitting of enamel" (FDI type 3; enamel pitting); transverse or linear enamel hypoplasia was

TABLE 1. Source and subdivisions of the study sample by taxon and sex[1]

Institution	Taxon	?	Female	Male	Total
Cleveland Museum of Natural History	*Gorilla*	8 (15.1)	19 (35.9)	26 (49.1)	53 (53.0)
	Pan	22 (55.0)	15 (37.5)	3 (7.5)	40 (40.0)
	Pongo	4 (57.1)	0 (0.0)	3 (42.9)	7 (7.0)
	Total	34 (34.0)	34 (34.0)	32 (32.0)	100 (73.0)
National Museum of Natural History	*Gorilla*	0 (0.0)	2 (33.3)	4 (66.7)	6 (16.7)
	Pan	6 (54.6)	2 (18.2)	3 (27.3)	11 (30.6)
	Pongo	1 (5.3)	11 (57.9)	7 (36.8)	19 (52.8)
	Total	7 (19.4)	15 (41.7)	14 (36.8)	36 (26.3)
	Grand total	41 (30.0)	49 (35.8)	46 (33.6)	n = 137

[1] One orangutan specimen from the personal collection of Rick Kellner is not included here except in the total n value.

absent from the deciduous dentition, 2) mandibular teeth were far more frequently affected by hypoplasia than maxillary teeth, 3) deciduous canines display hypoplastic lesions most frequently, "though this sample does not allow any very accurate estimate to be made of relative frequencies" (Eckhardt and Protsch von Zieten, 1993: 96), 4) labial or buccal surfaces were most commonly affected, and 5) the frequency of occurrence of enamel hypoplasia in this population ranges from 40% to 80%. What remains unclear is whether the "pitting" form of enamel hypoplasia described by Eckhardt and Protsch von Zieten is a small scale expression of the larger, oval and window-like, defects that Skinner refers to as LHPC, also accurately characterized as a "plane form defect" (Hillson and Bond, 1997). The data presented below expand upon results of this solitary prior study by using a larger and taxonomically more diverse sample of great apes, thereby allowing more accurate estimates of the relative frequency of enamel hypoplasia by individual and by tooth count methods.

METHODS AND MATERIALS

The specimens included in this analysis come from three sources: 1) the Hamann-Todd Collection at the Cleveland Museum of Natural History, 2) the Department of Mammals at the National Museum of Natural History, Smithsonian Institution, and 3) a personal specimen. The total sample consisted of 137 individual great ape specimens. Details regarding sample subdivisions by source institution, taxon, and sex are provided in Table 1. Approximately 69% of the specimens are accompanied by indication of sex, and the geographic source area for many individuals is known. Most individuals are represented by matching upper and lower jaws, though occasionally an isolated maxilla or mandible was scored. Any specimen that possessed one or more deciduous teeth was evaluated and all forms of enamel hypoplasia that were observed were recorded. In this sample 128 of the 137 specimens, or 93.4%, retained one or more deciduous canine teeth for observation of enamel hypoplasia.

Specimens were collected from their natural habitat during the turn of the century, a time when many museums sought to expand and diversify their inventory of non-human primate skeletons (Gregory and Raven, 1937; Jones-Kern and Latimer, 1996). Taxonomic identification to species or subspecies level was recorded for many, but not all, individuals studied. Because subdividing samples by both geographic and taxonomic (species) factors resulted in small sample sizes, analysis of enamel defect prevalence was conducted at the taxonomic level of genus. Though necessary for this preliminary statistical analysis, the lumping procedure clearly precludes precise characterization of interspecific variation of defect prevalence as well as detection of geographic differences within species. Despite these limitations, this analysis of intergeneric variation of enamel defects in the deciduous teeth of great apes has merit because: a) it reports a previously undescribed type of enamel defect; b) it reports it in a segment of the ape dentition (deciduous) not usually examined by dental anthropologists; c) it discovers significant variation in defect prevalence between ape genera; d) it thereby implies differential stress among ape taxa during early periods of ontogeny; and e) it will simulate further investigation into the prevalence and devel-

TABLE 2. Great ape skulls with localized hypoplasia of primary canine teeth (LHPC)[1]

	Female		Male		Total[2]	
Taxa	+/n	%	+/n	%	+/n	%[3]
Pan	4/17	23.5	1/6	16.7	11/50	22.0
Gorilla	18/20	90.0	25/27	92.6	47/53	88.7
Pongo	10/11	90.9	9/10	90.0	22/25	88.0
All apes	32/48	66.7	35/43	81.4	80/128	62.5

[1] One or more deciduous canine teeth scored + for LHPC.
[2] This column includes unsexed specimens; therefore, totals are greater than the sum of male and female values.
[3] A chi-square test of association revealed that differences in the frequency of enamel hypoplasia between taxa are statistically significant ($\chi^2 = 57.426$; $P = 0.000$).

opmental timing of enamel defects in the deciduous teeth of great apes.

Observations of enamel hypoplasias follow procedures recommended by Goodman and Rose (1990). Specimens were studied under oblique incandescent light, with secondary diffuse fluorescent background lighting. Initial observation was made with the naked eye, followed by closer inspection with a 10× power hand lens. Enamel hypoplasias were scored according to the standards recommended by the Federation Dentaire International (FDI) for evaluating Defects of Dental Enamel (DDE) (Federation Dentaire International, 1982; Clarkson, 1989), and use of the terms "pit" and "plane form" defect follow definitions of Hillson and Bond (1997). The size and location of enamel hypoplastic lesions was drawn on an outline diagram of the deciduous dentition. Individual defect height, breadth, and location on the crown were measured with a Helios needlepoint dial caliper calibrated to 0.05 mm. Analysis of the developmental timing of enamel hypoplasias based on defect position and size is now in progress and will be published soon, elsewhere.

RESULTS

Prevalence of enamel hypoplasia in three great ape genera: *Gorilla, Pan,* and *Pongo* are presented in two different formats. Table 2 provides data for the number of individuals with one or more canine teeth displaying enamel hypoplasia; this method of reporting is often referred to as the individual or specimen count method. The second report format, provided in Table 3, is known as the tooth count prevalence. This method reports the percentage of deciduous canine teeth with hypoplastic lesions as a percentage of all deciduous canine teeth observed. This is done by individual tooth, with summary statistics for maxillary and mandibular canines for each genus, and summations across all taxa for individual teeth. Photographs illustrating representative examples of LHPC in great apes from the Hamann-Todd Collection are provided in Figure 2.

While 62.5% of specimens exhibit LHPC in one or more canine teeth, this summary figure is misleading due to significant intergeneric variation in prevalence. The right hand columns in Table 2, labeled "Total," show that chimpanzees have significantly fewer individuals (22%) affected with LHPC than either gorillas or orangs ($\chi^2 = 57.43$; $P = 0.000$). Further, the prevalence of LHPC, by percentage of specimens affected, is high (88%) and nearly identical in orangutans and gorillas (Fig. 3a). A comparison of LHPC prevalence by sex reveals that both collectively, and by individual genus, sex differences are not significant. The tooth count prevalence of LHPC among great apes is presented in Table 3 and Figure 3b, while statistical values for chi-square tests are given in Table 4. Examination of these tables reveals that in all three great ape taxa, and for the composite sample, differences in LHPC prevalence between jaws is statistically significant, but none of the differences in prevalence between sides is significant. The difference in LHPC prevalence between right and left canines ranges from a low of 0.2% for the maxillary canines of *Pan,* to as high as 8.4% for chimpanzee mandibular canines (Fig. 3c). Composite values for all three taxa combined are provided in the right hand column of Table 3, where the difference between maxillary antimeres and mandibular antimeres is low (0.1%) and identical. Isomeric differences are another matter: they are great and consistently significant statistically (see Table 4). Mandibular canines exhibit LHPC from 1.5 to more than 5 times the rate for maxillary canine teeth. Though quantitative data on defect size will be reported later, mandibular canines have hypoplastic lesions that are more extensive than defects found in the maxillary canine teeth (refer to Fig. 1).

Another approach to the issue of LHPC expression by side in right and left anit-

TABLE 3. Prevalence of localized enamel hypoplasia (LHPC) in great ape canines by tooth count

	Pan		*Gorilla*		*Pongo*		Great ape total	
Tooth/jaw[1]	+/n	%	+/n	%	+/n	%	+/n	%
Urdc	2/39	5.1	21/42	50.0	5/18	27.8	28/99	28.3
Uldc	2/41	4.9	22/40	55.0	5/21	23.8	29/102	28.4
Maxilla	4/80	5.0	43/82	52.4	10/39	25.6	57/201	28.4
Lrdc	6/31	19.4	37/44	84.1	18/22	81.8	61/97	62.9
Lldc	10/36	27.8	34/41	82.9	19/23	82.6	63/100	63.0
Mandible	16/66	24.2	71/85	83.5	37/45	82.2	124/197	62.9
Total	20/146	13.7	114/167	68.3	47/84	55.9	181/398	45.5

[1] Urdc, upper right deciduous canine; Uldc, upper left deciduous canine; Lrdc, lower right deciduous canine; Lldc, lower left deciduous canine.

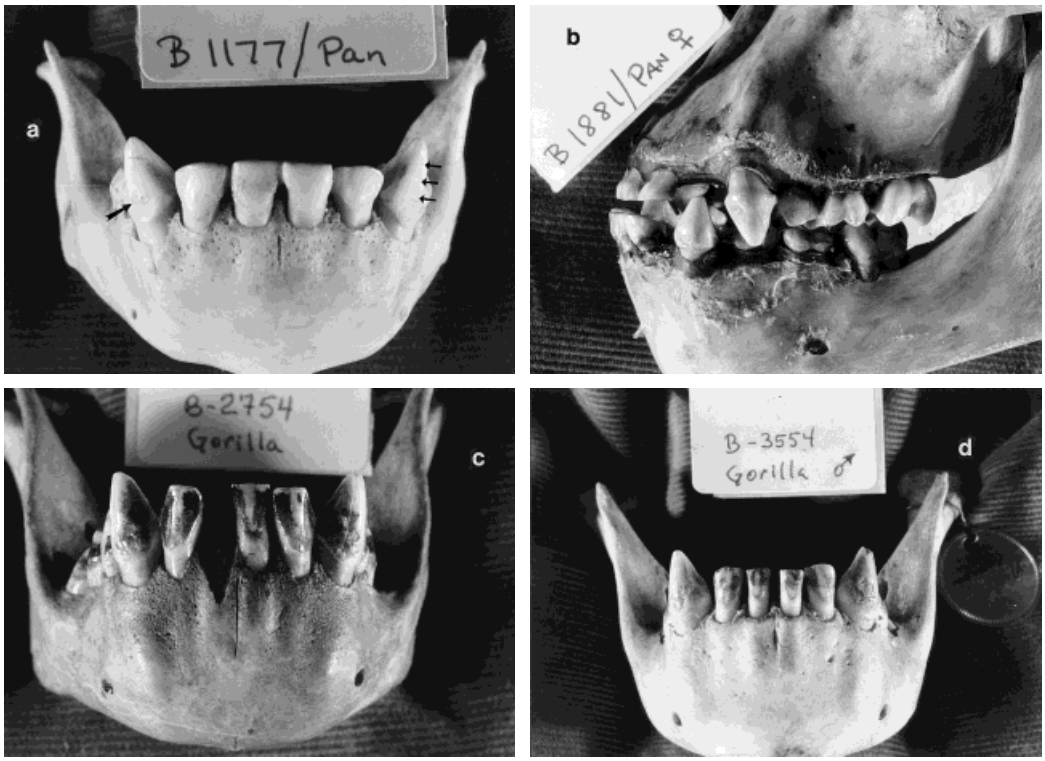

Fig. 2. Specimens from the Hamann-Todd Collection illustrating variation in the expression of enamel hypoplasia in the deciduous canine teeth of great apes. **a:** *Pan* number B-1177, mandible. The left canine displays multiple defects while the right has a large, severe defect near the cemento-enamel junction. **b:** *Pan* number B-1881, from Ebolwa, Cameroon. Left upper and lower primary canines exhibit characteristic enamel hypoplastic defects. **c:** *Gorilla* number B-2754, from Cameroon. Mandibular right and left deciduous canines display multiple "symmetrical" enamel hypoplasias. **d:** *Gorilla* number B-3554. Mandibular right and left deciduous canines display irregular enamel defects over the middle third of the labial surface of the crown.

meres is to use individual data. In this analysis each jaw of every specimen was classified into one of three categories of symmetry based on a comparison of right and left deciduous canine teeth. Since this analysis required the presence of both maxillary or both mandibular canine teeth, the sample sizes are somewhat reduced. Asymmetric positive individuals are those specimens in which one canine (right or left) was scored as hypoplastic and its antimere scored as not hypoplastic (LHPC-negative). Symmetric positive individuals display LHPC in both right and left canine teeth, and symmetric negative specimens have antimeres that were both scored as lacking LHPC. Maxillae

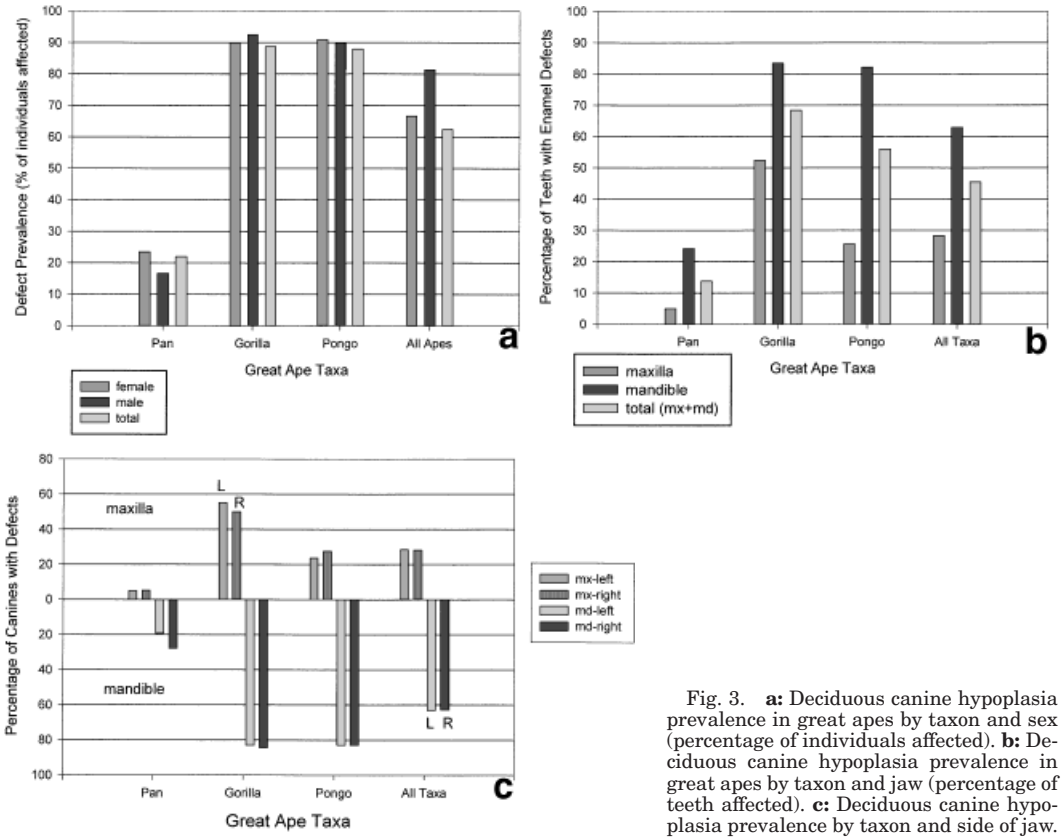

Fig. 3. **a:** Deciduous canine hypoplasia prevalence in great apes by taxon and sex (percentage of individuals affected). **b:** Deciduous canine hypoplasia prevalence in great apes by taxon and jaw (percentage of teeth affected). **c:** Deciduous canine hypoplasia prevalence by taxon and side of jaw.

TABLE 4. Variation in expression of LHPC by jaw and side of the dental arcade

	Jaw		Statistical values	
Taxon	Maxilla	Mandible	χ^2	*P*
Pan	5.0	24.2	11.33	0.000
Pongo	25.6	82.2	27.14	0.000
Gorilla	52.4	83.5	18.62	0.000
All taxa	28.8	62.9	47.99	0.000
	Side		Statistical values	
Taxon (jaw)	Right	Left	χ^2	*P*
Gorilla (max)	50.0	55.0	0.205	0.650
Gorilla (mand)	84.1	82.9	0.021	0.885
Gorilla (max + mand)	67.4	69.1	0.055	0.814
Pongo (max + mand)	57.5	54.6	0.074	0.785
Pan (max + mand)	11.4	15.6	0.539	0.463

TABLE 5. Prevalence of LHPC asymmetry and symmetry by jaw and taxon

Taxon	Asymmetric +	Symmetric +	Symmetric −	Total
Maxilla				
Gorilla	7 (18.9)	16 (43.2)	14 (37.8)	37 (41.6)
Pan	2 (5.9)	0 (0.0)	32 (94.1)	34 (38.2)
Pongo	3 (16.7)	3 (16.7)	12 (66.7)	18 (20.2)
Total	12 (13.5)	19 (21.4)	58 (65.2)	89 (100.0)
Mandible				
Gorilla	5 (13.5)	29 (78.4)	3 (8.1)	37 (43.0)
Pan	1 (3.6)	6 (21.4)	21 (75.0)	28 (32.6)
Pongo	5 (23.8)	15 (71.4)	1 (4.8)	21 (24.4)
Total	11 (12.8)	50 (58.1)	25 (29.1)	86 (100.0)
Grand total	23 (13.1)	69 (39.4)	83 (47.4)	175 (100.0)

and mandibulae were evaluated separately, and reveal some valuable insights.

Scrutiny of the data in Table 5 reveals several important aspects of LHPC distribution. First, the grand total values (bottom line), across all taxa and for maxilla and mandible combined, show that symmetry (86.8%), whether positive or negative for LHPC, is much more common than asymmetry (13.1%). Second, the percentage of asymmetric positive specimens is roughly equal for the maxilla and the mandible of all taxa, and *Pan* has a lower frequency of asymmetric positive individuals than either *Gorilla* or *Pongo.* Third, the prevalence of positive symmetry is significantly greater in the

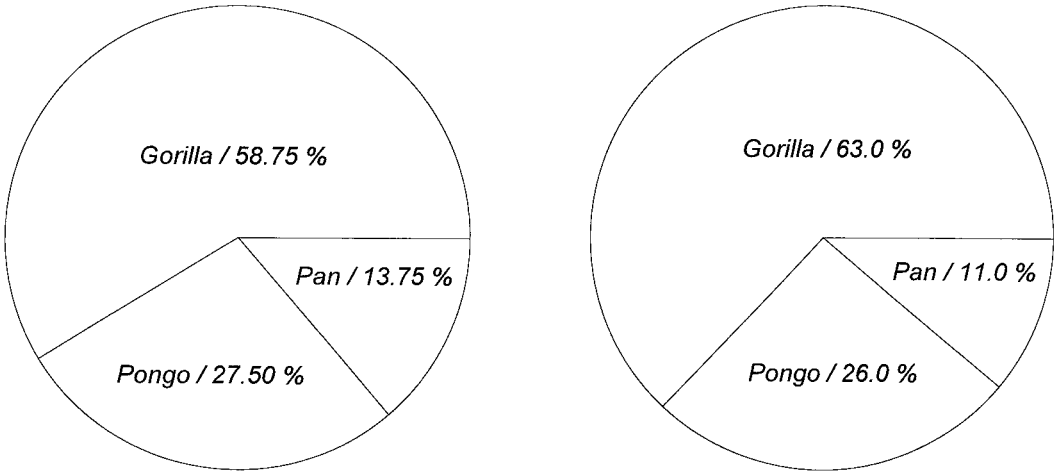

Fig. 4. Percentage of affected individuals by taxon (left) and percentage of affected canine teeth by taxon (right).

mandible than in the maxilla of all three taxa. Finally, *Gorilla* displays a significantly higher incidence of symmetrically positive jaws than either chimpanzees or orangutans. These differences in symmetry between jaws and taxa may provide clues regarding the possibility of intergeneric differences in the proximate etiology of LHPC.

While the expression of canine hypoplasia is variable in great apes and in humans, there are some basic similarities and differences in defect appearance that are noteworthy. In humans the most common form of expression is a roughly oval or circular "plane form" defect. Discontinuous double or multiple defects may occur but they are discrete and clearly localized (see Lukacs and Walimbe, 1998, Figs. 4, 5). In great apes some defects exhibit the human form of expression (see Fig. 1). Much more common among great apes are multiple small (0.5–2.0 mm) "plane form" defects of circular or irregular oval shape that may be discrete or confluent (Fig. 2). One possible variable contributing to this difference in defect expression between apes and humans is enamel thickness of deciduous canines. Enamel thickness on the facial (labial) surface of human deciduous canine teeth appears to be greater than in great ape deciduous canines. The fact that in human deciduous teeth mandibular canines display thicker enamel than maxillary canines may contribute to significant differences in defect prevalence and size (Rahn et al., 1997). No corresponding quantitative data are currently available for great ape deciduous canines; however, the great apes included in this study appeared to have thinner enamel than human samples. Variation in enamel thickness of deciduous teeth of hominoids requires further comparative study since this variable may have significant implications for hypoplastic defect expression.

As a final, graphic display of results, Figure 4 presents two pie charts that illustrate the breakdown of affected individuals (left) and affected canine teeth (right) by genus. Slices of the pie represent the percentage of all affected specimens (or all hypoplastic teeth) that are LHPC positive for each genus. The size of each slice is a function of the number of affected specimens or teeth in a genus relative to all affected specimens or teeth; it is not a function of the size of study samples. A methodological issue that often concerns researchers focusing on meristic or repeating characters is, how closely similar, or widely divergent, are percentages based on the number of individuals affected with a condition and percentages based on affected parts of individuals? In this case, the close similarity of the two pie charts, the segments of which differ by only a few percentage points, reveals that in this study sample there is indeed a very close correspondence

between the results obtained from individual counts and from tooth counts. This is not universally the outcome of such studies and caution must be exercised to test each study sample for agreement.

DISCUSSION

Do significant differences in defect prevalence imply differential levels of physiological stress in these three genera of great apes? Are chimpanzees truly less subject to developmental stress than gorillas and orangutans, as the differences in frequency of LHPC suggest? These are difficult questions to answer for several reasons: 1) the etiology of localized hypoplasia of primary canine teeth (LHPC) in human populations is not fully understood, 2) this analysis includes intergeneric comparisons, and therefore differs from intraspecific comparisons of defect prevalence within *Homo sapiens,* and 3) the expression of LHPC may result from multiple etiological influences, thereby reflecting a mixture of species-specific differences in morphology and development as well as providing an indirect measure of physiological stress. While several investigators seem to accept enamel hypoplasia of primary canine teeth as a retrospective but direct measure of physiological or developmental stress (in Liberian chimpanzees, by Eckhardt and Protsch von Zieten, 1993; in prehistoric humans in India, by Lukacs and Walimbe, 1998, 1999; in late Pleistocene hominines, by Skinner, 1996), the relationship may not be as direct or clear-cut as investigators assume.

LHPC prevalence in apes and humans

One approach to interpreting intergeneric variation in LHPC among great apes is to use modern humans as a model. Close genetic relationship between apes and humans, and similarities in LEH prevalence among hominoids, justifies using human LHPC prevalence data to help understand variation among great apes. In human populations, prevalence of LHPC shows significant positive associations with ethnic group, socioeconomic status, nutritional status and climate induced subsistence change (see Lukacs and Walimbe, 1998, 1999). Nutritionally and economically disadvantaged groups consistently show higher frequencies of LHPC than groups whose health, nutrition, and economic status is at or above the "norm" or "average." This finding is true for clinical and for epidemiological studies, and applies with equal force to children from distinct geographic locales and from diverse ethnic backgrounds. Without complete knowledge of the etiological pathway for LHPC, the clear association of high prevalence with stressful environments suggests a causal yet complex relationship (Lukacs, 1991).

In a study of LHPC prevalence and etiology among Canadian children in the Healthiest Babies Possible Program (Skinner et al., 1994), clear associations were found by Skinner and colleagues between the presence of LHPC and ethnicity, nutrition, and birth month. Important results of their analysis of LHPC etiology include the following observations: 1) that it is the most common form of enamel hypoplasia in the deciduous dentition, 2) that it has higher prevalence in nutritionally disadvantaged study groups, 3) that more severe defects and higher rates of occurrence are evident in people of Asian Indian heritage, 4) that children born during months of lower sunlight have higher rates of the defect, and 5) the most significant nutritional factor among affected individuals is lower levels of retinol (vitamin A). These observations on human populations might encourage us to interpret differential prevalence of LHPC in great apes as a direct indicator of physiological stress. The logical and sensible conclusion from the data presented here is that gorillas and orangutans are more heavily influenced by developmental stress during early stages of ontogeny than chimpanzees. However, a direct linear relationship between defect expression or prevalence and stress level or severity is inappropriate (Hillson and Bond, 1997), especially when multiple morphologically diverse taxa are the subject of study. The analysis of great ape LHPC involves intergeneric comparisons, consequently morphological and developmental differences among the genera being compared may contribute to defect prevalence and expression. Defect prevalence and expression is interpreted to reflect the combined influence of level of physiological stress and group specific variations in morphology and development.

TABLE 6. Mean crown diameters of great ape deciduous canine teeth (in mm)

	Mesio-distal "Length"				Bucco-lingual "Breadth"			
	Ashton and Zuckerman (1950)			Greenfield (nd)[1]	Ashton and Zuckerman (1950)			Greenfield (nd)
	n	$\bar{x}$	sd	$\bar{x}$	n	$\bar{x}$	sd	$\bar{x}$
Maxilla								
Chimp	16	7.9	0.8	7.9	16	5.7	0.7	5.5
Orang	13	9.3	0.7	8.9	13	7.2	0.7	6.3
Gorilla	17	10.7	1.0	9.7	16	8.0	1.9	7.5
Mandible								
Chimp	15	6.9	1.1	7.3	15	6.2	0.5	5.8
Orang	9	8.5	0.9	8.2	8	6.9	0.7	6.9
Gorilla	13	9.0	0.9	8.1	12	7.0	0.9	6.5

[1] Unpublished data.

Factors influencing the expression of LHPC

The phenotypic expression of any biological character is the outcome of a complex series of interactions between genetic and environmental forces. Therefore, the possibility must be considered that there are aspects of genetics, morphology, and development that predispose one taxon to more easily develop canine enamel hypoplasia than another. And, that variation in these same factors may render the genesis of LHPC less likely in another taxon, given the same level of physiological stress. For example, variation in the size of deciduous canine teeth, shape of the developing jaw in the canine region, and timing of deciduous dental development are factors that may vary significantly between taxa and may directly impact the probability of forming enamel defects in canine teeth. These ideas are best regarded as tentative hypotheses that are described here to stimulate further research into the morphology, timing, and development of great ape deciduous teeth. However, some preliminary data on tooth size and dental calcification may be cited as relevant evidence of such intergeneric variation, but clearly further research, including allometric approaches are required.

For example, crown dimensions of great ape deciduous teeth show considerable variability. Though odontometric data for great ape deciduous teeth are limited, the two available sources provide a consistent picture of variation in size (Table 6; Ashton and Zuckerman, 1950; Greenfield, personal communication). The average difference between mean values for mesiodistal (MD) crown diameters and for buccolingual (BL) crown diameters reported by these two investigators is less than 0.5 mm, suggesting consistent observations by researchers studying different samples and undoubtedly using subtly different measurement techniques. The data in Table 6 reveal that chimpanzees have significantly smaller deciduous canine crown diameters than either the gorilla or the orangutan. In explaining variation in LHPC prevalence between taxa, consideration must be given to the fact that teeth are developing in a spatially constricted and rapidly changing environment—the deciduous dental crypt. It may be that large teeth encounter space limitations that affect enamel formation more frequently than small teeth. When this is combined with the fact that the canine crypt is situated in the region of maximal curvature of the jaw, it predisposes to problems of restricted space and promotes contact between the developing germ and the crypt wall that may injure or retard the productivity of ameloblasts. Enamel defects of deciduous canine teeth undoubtedly have a complex and multifactorial etiology that reflects levels of physiological stress as well as a variety of confounding genetic, developmental, morphological, and environmental factors.

The inference of intergeneric differences in physiological stress among great apes is derived from significant variation among taxa in LHPC prevalence. The developmental timing of enamel defect formation among great apes is a separate, though relevant, issue for which data are largely unavailable. Inability to provide specific data regarding differences in timing of defect formation among great ape taxa does not diminish the

insights gained from prevalence data. While data on calcification of deciduous teeth among the great apes are elusive, several reports are available on chimpanzee perinatal dental development (Siebert and Swindler, 1991; Tarrant and Swindler, 1972). These data reveal that deciduous canines lag behind incisor teeth in calcification. Prenatally, at about the fifth to sixth fetal month, chimpanzee deciduous canines displayed a partially calcified crown covering a soft template. At full term, only the tip of the canine is calcified, while a 4-month-old female chimpanzee had a calcified canine crown. Small samples are a limiting factor in these studies, but provide basic data for chimpanzees: calcification of the deciduous canine begins at about the fifth fetal month and is completed by the fifth month of postnatal development. The timing of canine enamel defects among chimpanzees would be shortly before and just after birth, reflecting physiological stress during this segment of perinatal development.

In addition to factors of tooth crown size, crypt space, jaw curvature, and calcification, other developmental and genetic variables may contribute to the differences in enamel hypoplasia prevalence observed in this study. Although multiple, and possibly different, etiological factors are likely to contribute to LHPC prevalence in these taxa, one of these factors is physiological stress or growth disruption. By analogy with humans, the higher prevalence of enamel defects in the deciduous teeth of gorillas and orangutans suggests that levels of developmental stress may be significantly greater in these great apes than in chimpanzees. The corollary is that chimpanzees are relatively less stressed than their larger-bodied relatives during early stages of ontogeny.

These data on prevalence and expression of enamel defects in the deciduous canine teeth of great apes provide a valuable comparative perspective for understanding the proximate etiology of LHPC in human populations. In 1986, Skinner (1986a:64–65) stated, "Furthermore, casual observation that the defect occurs in the canines from precontact Native Americans and chimpanzees indicates that the cause of the defect is not exclusive to human beings or to particular cultures, geographic areas, or time periods."

To this preliminary observation new insights derived from the present study may now be added: the pattern of LHPC distribution in great apes closely follows the pattern of defect distribution in humans—significant differences in prevalence occur between jaws, and significant differences are absent when right and left sides of the arcade are compared. However, there are some impressionistic differences in jaw structure and enamel thickness (discussed above) that deserve further investigation. The mandibular cortical bone overlying the canine crypt was judged to be robust and thick in all great ape taxa in this study. By contrast, in humans the condition is quite different, with thin and often discontinuous cortical bone—especially over the mandibular canine—exposing the labial wall of the developing canine. This condition has been implicated in the etiology of LHPC in humans (Skinner and Hung, 1989, Fig. 6). The idea that exploratory "mouthing behavior" may be involved in defect formation in both apes and human, though possible, seems highly unlikely (Skinner and Hung, 1989:171, Fig. 12). More details regarding the progress of calcification in great ape deciduous teeth, the relationship of dental development to behavioral patterns and to nutritional status will be required to fully understand the meaning of intergeneric variation in enamel hypoplasia of deciduous teeth in extant hominoids.

Prevalence of enamel defects in deciduous (LHPC) and permanent (LEH) teeth

How does the frequency of enamel defects in deciduous canine teeth compare with the prevalence of LEH in permanent teeth of great apes? The prevalence of enamel hypoplasia in the permanent teeth of chimpanzees and gorillas from the same skeletal collection at the Cleveland Museum of Natural History was recently reported by Stottlemire (1998). A dramatic dichotomy in frequency of enamel defects was found in permanent teeth, with chimpanzees (80.6% of individuals, n = 98) about three times more frequently affected than gorillas (27.5%, n = 229). In contrast to the results

reported here for the deciduous canine teeth of these taxa (chimps, 22%, n = 50; gorillas, 88.7%, n = 53), Stottlemire's findings are similar in degree, but taxonomically reversed. The higher prevalence of enamel hypoplasias in the deciduous teeth of gorillas and in the permanent teeth of chimpanzees could reflect: 1) stress during different stages of development in these two taxa, with greater stress during perinatal and infant development in gorillas, but in later childhood among chimpanzees, 2) different etiological factors in the production of LEH defects in permanent teeth vs. localized enamel hypoplasia of deciduous canine teeth, or 3) interobserver differences methodology. Also, noteworthy is the lack of agreement between Stottlemire's (1998) results and the findings reported by Skinner (1986b) for LEH among chimpanzees and gorillas from the Cotton-Powell Museum. In Skinner's study, gorillas were found to have a significantly higher incidence of LEH than a sympatric sample of chimpanzees. These results raise several questions that deserve further investigation: Does the expression of LHPC (deciduous teeth) and LEH (permanent teeth) within individuals reveal a positive or negative association? Does susceptibility to enamel hypoplasia early in life predispose an individual to systemic stress later in life? How much variation in LHPC and in LEH prevalence exists between study samples from different museums, ultimately drawn from different ecological and environmental settings?

CONCLUSIONS

This preliminary investigation of enamel hypoplasias in deciduous canine teeth of three genera of hominoids has contributed significant new insight into differential physiological stress among extant hominoid taxa during early stages of ontogeny.

1) Linear enamel hypoplasia is absent from the deciduous teeth of great apes, but lesions phenotypically similar to localized hypoplasia of primary canines (Skinner, 1986a) are commonly found on great ape deciduous canines.

2) Overall 63% of specimens display LHPC on one or more deciduous canine, but the individual count prevalence among taxa shows significant variability: chimpanzees (22%), gorillas (89%), and orangutans (88%).

3) The pattern of defect distribution by jaw (maxilla vs. mandible; significant differences) and side (right vs. left; no significant differences) reveals close similarity to the pattern of defect distribution in human populations.

4) Etiological analogy with human populations, where nutritionally and economically disadvantaged population have higher rates of enamel defects in their deciduous teeth, suggests that gorilla and orangutan samples included in this study suffered significantly more physiological stress during early development than chimpanzee samples. Further analysis of larger samples from local natural populations are essential to determine if enamel defect types and prevalences are systematically patterned or random.

5) The pattern of enamel defect distribution in apes and humans, when combined with further research into deciduous dental calcification, timing of deciduous dental development, behavioral patterns, and nutritional status, will lead to a better understanding of the etiology of these enigmatic lesions.

ACKNOWLEDGMENTS

For access to specimens and logistical assistance in completing this research thanks are due to Dr. Bruce Latimer and Lyman Jellema, Laboratory of Physical Anthropology, Cleveland Museum of Natural History, and to Dr. Richard Thorington and Linda Gordon, Department of Mammals, National Museum of Natural History (Smithsonian Institution). Travel and research funds were provided by a faculty research grant from the University of Oregon, Department of Anthropology.

Special thanks are due to Rick Kellner, BS Anthropology, University of Oregon, who provided the juvenile orangutan skull that stimulated this study, to Debbie Guatelli-Steinberg, PhD Biological Anthropology (1998), for discussions on primate enamel defects and stress levels, and the many colleagues who promptly replied to my pleas for sources, data, and commentary (Len Greenfield, Lynn Kilgore, Kevin Kuykendall, Nancy Lovell, Daris Swindler, Linda Winkler).

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